Perspective

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Reproductive Endocrine Hormones in Males and Females

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Description

For anti-doping and clinical Point Of Care (POC) analysis, there is a significant demand for the development of screening methods that are both rapid and high-throughput. It has been demonstrated that target analysis in complex sample matrices can be accomplished quickly and effectively using solid-phase micro extraction and mass spectrometry direct coupling. In this work, a polymer-coated open-tubular column was used to develop an online direct coupling of in-tube SPME with MS. After elution from the IT-SPME column, a sharp stainless-steel needle was attached to the end of the SPME column, allowing for the direct ionization of the analytes. As the extraction phase, itaconic acid-benzene co-polymer was grown in situ on the inner surface of the fused silica capillary. Due to its chemical properties, this column offers both hydrophobic and weak cationic exchange interaction with the target analytes. The developed online IT-SPME-MS method was able to be used at least 60 times and performed well when it came to extracting various target analytes. The above method was used as a proof-of-concept for the analysis of antiepileptic drugs in plasma and urine with a linear range of 1 ng/mL to 200 ng/mL, good linearity and good reproducibility. For the two AEDs, the method had enrichment factors between 187 and 204 and high sensitivity for analyzing human plasma and urine samples.

Hormonal Changes

Epilepsy is a chronic brain disorder that is not contagious and affects men and women of all ages. It is characterized by recurrent seizures accompanied by brief episodes of sensory, motor, or autonomic events, with or without consciousness. It is one of the most prevalent neurological conditions, affecting approximately 50 million people worldwide. It necessitates long-term treatment with antiepileptic drugs in some cases. The use of AEDs for an extended period of time has been linked to an increased risk of fracture, abnormal bone development, changes in calcium metabolism, and hormonal changes. The various metabolic reactions are controlled by hormones, which are various molecules. It is well documented that both male and female reproductive endocrine hormones are affected by chronic AED therapy. The multifactorial nature of AED-induced bone loss includes: estrogen deficiency, calcitonin deficiency, secondary hyperparathyroidism, and hypovitaminosis D. Although some newer antiepileptic drugs have also been reported to cause bone loss, enzymes that induce AED metabolism primarily cause bone loss through the induction of vitamin D catabolism and reduction in the level of biologically active sex steroid hormone. In AED-induced bone loss, the precise mechanisms of estrogen deficiency and TGF-mediated bone turnover are currently unclear. The determination of bone architecture is frequently accomplished by measuring the organization of bone cellular structure, cortical bone thickness, and trabecular bone volume. Estrogen, parathyroid hormone, and the Transforming Growth Factor (TGF) signaling pathway control bone mass and architecture; dysregulation of these pathways alters bone turnover, which causes osteoporosis. Transforming growth factor a cytokine superfamily member, is a polypeptide with multiple physiological functions, including regulating bone mineral density. As a result, it could be investigated as a potential indicator of osteoporosis or bone loss. This review focuses primarily on highlighting the possibility that TGF- and estrogen deficiency play a role in AED-induced bone loss. An effort has been made to advance our understanding of the underlying pathophysiological processes and the mechanism by which AEDs cause bone loss; with a special focus on the roles that TGF and estrogen deficiency play in the bone loss caused by AEDs. As a result, it is anticipated that they could be used as a potential indicator of bone turnover in patients receiving chronic AED therapy, facilitating the diagnosis and treatment of chronic epilepsy by researchers and clinicians.

Drug Effects on Axonal Elongation

It has been hypothesized that children born to pregnant women who take 1000 mg or more of valproic acid per day have a lower intelligence quotient than children born to pregnant women who take other antiepileptic medications. However, the reason why children exposed to valproic acid during the fetal period have a lower intelligence quotient is still unknown. To assess the effects of valproic acid-containing antiepileptic medications on nerve cells, we utilized the human neuroblastoma cell line SH-SY5Y. Using the Cell Counting Kit-8, we carried out a WST-8 colorimetric assay to examine the drugs' anti-proliferative effects in these cells. Using image software, we also quantified drug effects on axonal elongation. Using real-time PCR, we also looked at how drugs affected the levels of mRNA expression on molecules involved in the development of

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the nervous system and uptake of folic acid. At concentrations greater than 500 M, carbamazepine and lamotrigen were toxic to SH-SY5Y cells, whereas phenytoin and valproic acid were not. Axonal outgrowth was unaffected by phenytoin, lamotrigen, valproic acid, and carbamazepine in SH-SY5Y cells. When valproic acid was added to the cells, the ratios of the mRNA expression levels of sodium channel neuronal type 1a (SCN1A) increased. Neurodevelopment may be harmed by SCN1A mRNA overexpression caused by high valproic acid concentrations in the fetus. However, because specific mechanisms have not yet been discovered, it is necessary to compare cell axon elongation and SCN1A protein expression after high-concentration valproic acid exposure to assess the effect. For brain disease research and drug evaluation, brain organoids with three-dimensional structure and tissue-like function are highly sought after. However, to our knowledge, there have not yet been developed methods for measuring and analyzing brain organoid function. This study evaluated the response to the antiepileptic drugs perampanel and phenytoin as well as the convulsants pentylenetetrazol and strychnine in a waveform obtained below 500 Hz using a planner microelectrode array (MEA). PTZ administration resulted in sudden and persistent firing resembling seizures, exhibiting a concentration-dependent periodic activity with an enhanced frequency component even in one oscillation characteristic. However, when AEDs were administered, both the intensity of a single oscillation's frequency component and its frequency frequency decreased concentration-dependently. A group of synchronized bursts was observed at low phenytoin doses, which was distinct from the perampanel response. MEA was found to be useful in predicting the seizure liability of drugs and evaluating the effect of AEDs with a different mechanism of action because frequency components contained information on cerebral organoid function. In addition, MEA-based frequency component analysis of brain organoids is a crucial method for future *in vitro* to *in vivo* extrapolation, which will aid in the investigation of the organoid's function, the study of human brain development and the treatment of various brain diseases.